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Evaluation of Five Diets on the Occurrence of Spring Thing

by

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ABSTRACT

An unknown disease has afflicted summer chinook salmon fry at McCall Hatchery since operations began in 1980. Known as Spring Thing (ST), it has been responsible for mortality rates at McCall as high as 27%.

In 1984, swim-up fry were tested using five different feed rations and evaluated for mortality, growth, body condition and cost effectiveness. Moribund and healthy fish were examined histologically during the study.

Results of the 100-day study showed all test groups contracted ST. Mortalities in all test groups were lower than those experienced in past years and one diet, OP-4 + ten times the normal amount of pantothenic acid (OP-4 acid), had the lowest mortality of all groups (0.77%).

Fish on OP-4 acid had the highest percent weight gain per fish (173.7%) and also produced fish with the highest condition factor. Throughout the study, fish fed OP-4 acid had an increased feeding response and were healthier looking than the other test fish. The lowest cost per pound of fish produced was obtained with the OP-4 + four times the vitamin premix diet ($2.16).

Histologically, tissues of moribund fish exhibited abnormal pathological changes including hyperplasia of gill epithelium and degeneration of liver cells and kidney tubules.

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INTRODUCTION

Construction of hydroelectric dams on the Columbia and Snake rivers has reduced Idaho's chinook salmon runs to critically low levels. The McCall Summer Chinook Salmon Hatchery was constructed in 1979 as partial mitigation for losses of chinook caused by the Lower Snake River dams (Ice Harbor, Lower Monumental, Little Goose and Lower Granite). Its objective is to help restore summer chinook salmon to the South Fork Salmon River (SFSR). Historically, the SFSR was the major summer chinook spawning stream in Idaho, supporting as many as 20,000-30,000 adults (Idaho Anadromous Fish Management Plan 1984-1990).

During the first year of hatchery production (1980) an unknown mortality syndrome appeared in the chinook fry. Affected fish exhibited lethargic behavior, side-swimming, some spiraling along the long axis and poor feeding response (Wimer 1980). Examination of the gills showed hyperplasia of lamellae and in many cases, fusion of filaments. Externally, the ventral profile of fry showed a characteristic pinched-appearance anterior to the vent (Fig. 1). These clinical signs ultimately led to mortality of 24% of the fish. The disease appeared to be similar to clubbed-gills-"dropout" disease described by Wood (1979). However, affected fish did not always fall under the category of "pinheads" (Fig. 2). Instead, they were apparently healthy fish having good body condition (Fig. 3).

A similar mortality pattern occurred in 1981 and was responsible for the death of 26% of the fish. For lack of a specific name and because the mortalities appeared in the spring, the disease syndrome was termed Spring Thing (Hutchinson 1981). Pathologists from Idaho, Montana, Utah and Washington were involved in an attempt to find a causal agent but none was found.

In 1981, the possibility of a nutritional deficiency being responsible for ST was discussed. It was decided to test several commercial feeds on fry from the upcoming brood year and evaluate their performance with regard to survival, growth and dietary efficiency against the standard chinook diet, Oregon Moist Pellet II (OP-2). The feeds tested did not prevent the outbreak of ST, but one diet (OP-4) outperformed all others (Thorpe and Hutchinson 1983). OP-4 is a variation of the OP-2 diet but contains no cottonseed meal and has a higher fish meal protein (47% vs. 28% in OP-2).

During 1982-83 another feed study was conducted but had to be terminated due to the feed manufacturer's inability to supply the various diets. However, mortality rates of production fish on OP-2 and OP-4 were compared; OP-4 sustained a 17% mortality while OP-2 had 26%.

In 1983-84, another group of diets were tested with the hope of reducing our spring mortality and evaluating feed performance. Since hyperplasia of gill epithelium has been nutritionally related to pantothenic acid deficiency (Wolf 1945, Rucker et al. 1952, Halver 1972 and Snieszko 1974) one of the test diets contained increased levels of pantothenic acid. We were hopeful this study would finally shed some light on a recurring problem.

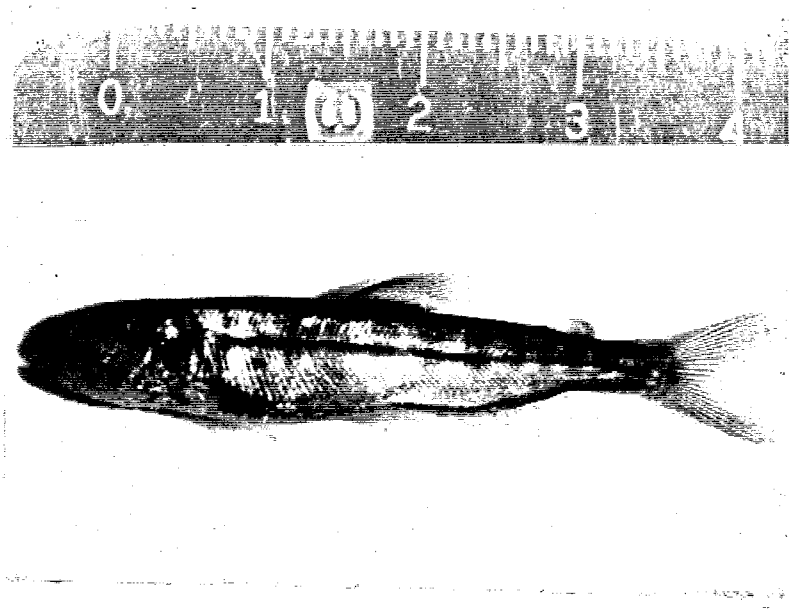


Figure 1. Photograph of a fish with Spring Thing. →

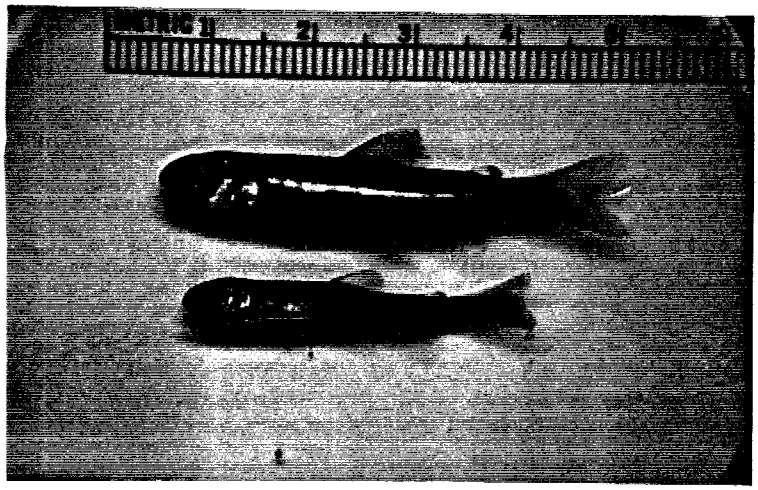


Figure 2. Photograph of fish with Spring Thing (top) and a typical "pinhead" (bottom). ←

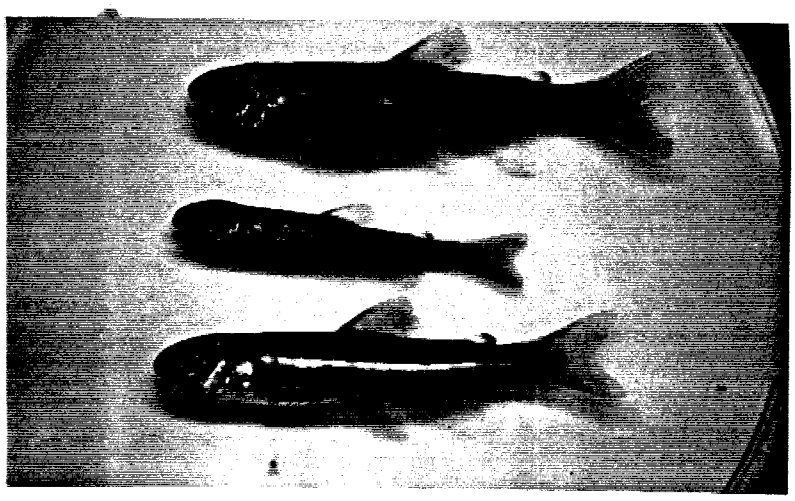


Figure 3. Photograph of a normal fish (top) "pinhead" (middle) and a fish with Spring Thing (bottom). →

OBJECTIVES

Test the efficiency of five different rations in the elimination of reduction of mortality associated with ST.

Evaluate the five different rations with regard to growth, body condition and cost effectiveness.

Evaluate fish health from a histological/histopathological standpoint during the study.

Determine vitamin levels of each ration during a four-month period by laboratory analysis.

METHODS

SFSR summer chinook fry from the 1983 brood year were used in the 100-day nutrition study. Because of low adult returns, eggs from three different egg takes were used: 14 taken on 18 August, #5 taken on 22 August, and #7 taken on 29 August. Eggs were incubated in Heath trays and after accumulating 1,600 $\pm$ 5 temperature units, fry were transferred to five rectangular tanks (4^1 x 40^1 x 2.5^1). Fry from egg take 14 were started on feed 9 December, #5 started on 22 December and #7 on 17 January. Study groups were reared under standard production conditions, and each tank contained approximately 81,000 fry.

Fish densities were maintained at or below the Maximum Density Index (Klontz 1979) with flows ranging from 3.4-4.7 turnovers per hour. Water temperature during the study period averaged 38± 1 F. Tanks were cleaned daily and any mortalities were removed and recorded.

Five diets were tested: OP-4, OP-4 plus ten times the normal amount of pantothenic acid (OP-4 acid), OP-4 plus four times the normal amount of vitamin premix (OP-4 vit.), OP-4 that had been vacuumed packaged (OP-4 vac.) and equal amounts of OP-4 and processed beef liver (OP-4 liver). All test diets except OP-4 liver were prepared by Bioproducts, Inc., Warrenton, Oregon. The OP-4 liver diet was prepared at the hatchery by mixing equal portions of OP-4 and processed beef liver.

Representative samples of fish were weighed and counted on the 1st and 15th of each month, and daily feed requirements were calculated on a percent-body-weight basis. Initially, all test groups were fed at levels of 6% total body weight per day then reduced to 3% when fish reached approximately 800 fish per pound. Each group was fed Oregon Moist Starter Mash for the first two days on feed. For the next five days they were fed a 50/50 mix of starter mash and their designated diet (size 1/32 pellet). For the remainder of the study, fish were fed only their assigned ration. Half of the daily feed requirement was weighed out at 0800 and 1300 hours and fish were fed by hand ten times per day.

Fish samples were collected on 7, 14 and 29 March and 10 April for histological examination. Ten fish from each group were collected from the head, middle and tail portions of the rearing tanks. Five fish from each group were immediately fixed in Boiuin's solution. The remaining fish were subjected to microscopic examination of the gills and gut and searched for internal and external parasites.

Samples were prepared for sectioning using routine histological techniques. Both longitudinal sections and pooled cross-sections were cut at 5-7 micrometers thickness. In cases where fish exhibited clinical signs of ST, gills were pooled and sectioned. In some cases, the gastro-intestinal tract was removed and sectioned separately.

Sectioned samples were fixed on micro-slides and stained with Hematoxylin and Eosin. In some cases, May-Grunwald Giemsa stains were used. Prepared slides were microscopically examined with emphasis given to the following organs/tissues: gills, gut, kidney, liver, swim-bladder and pancreatic tissue.

All five diets were analyzed for eight different vitamins by Blomed Research Laboratories, Seattle, Washington. Twelve-ounce samples of each diet were placed in an insulated container with dry ice and shipped for analysis. Samples were collected at five intervals from 5 December to 29 March.

RESULTS

Fish in all test groups suffered mortalities from ST. Mortalities resulting from this disease first appeared in mid-March and continued through the conclusion of the study. The OP-4 acid diet had the lowest mortality rate (0.77%) while the OP-4 vac. had the highest (1.91%) during the 100-day study (Table 1). Mortality in the OP-4 acid group remained consistently lower than all other diets throughout the evaluation period (Fig. 4).

The test group on OP-4 acid also had the highest percent weight gain per fish (173.7%) while the other diets ranged from 72.6% through 147.9% (Table 1). Fish on OP-4 acid had the highest condition factor (C = ratio of fish weight to the length cubed) at the end of the study, $C = 3504 \times 10'^{7}$. In addition, the OP-4 acid fish were healthier looking throughout the study and had an increased feeding response over all other test groups.

Cost effectiveness is an important consideration in evaluating performance at a production hatchery. The lowest cost per pound of fish produced was attained in the OP-4 vit. group ($2.16) while the OP-4 acid group was second ($2.25).

Moribund and clinically healthy fish were examined histologically by Charlie Smith, Fish Cultural Development Center, Bozeman, Montana, and Dr. A. Jim Chacko, University of Idaho, Moscow, Idaho. Gross pathological changes in gills were noted in many of the moribund and

Table 1. Nutrition study data comparing five diets for 100 days.

Diet	Fish/lb at start	Fish/lb at end	Percent gain	% Mortality	Condition factor*	Cost/lb fish produced
OP-4	1,022.9	562.5	81.8	1.70	2915	$ 3.20
OP-4 acid	1,135.0	414.7	173.7	0.77	3504	2.25
OP-4 vit	1,061.7	523.9	102.7	1.50	3069	2.19
OP-4 vac	1,022.9	592.8	72.6	1.91	2646	4.09
OP-4 liver	1,061.7	428.3	147.9	1.19	3115	6.12

* $C = Weight/Length^3 \times 10^{-7}$

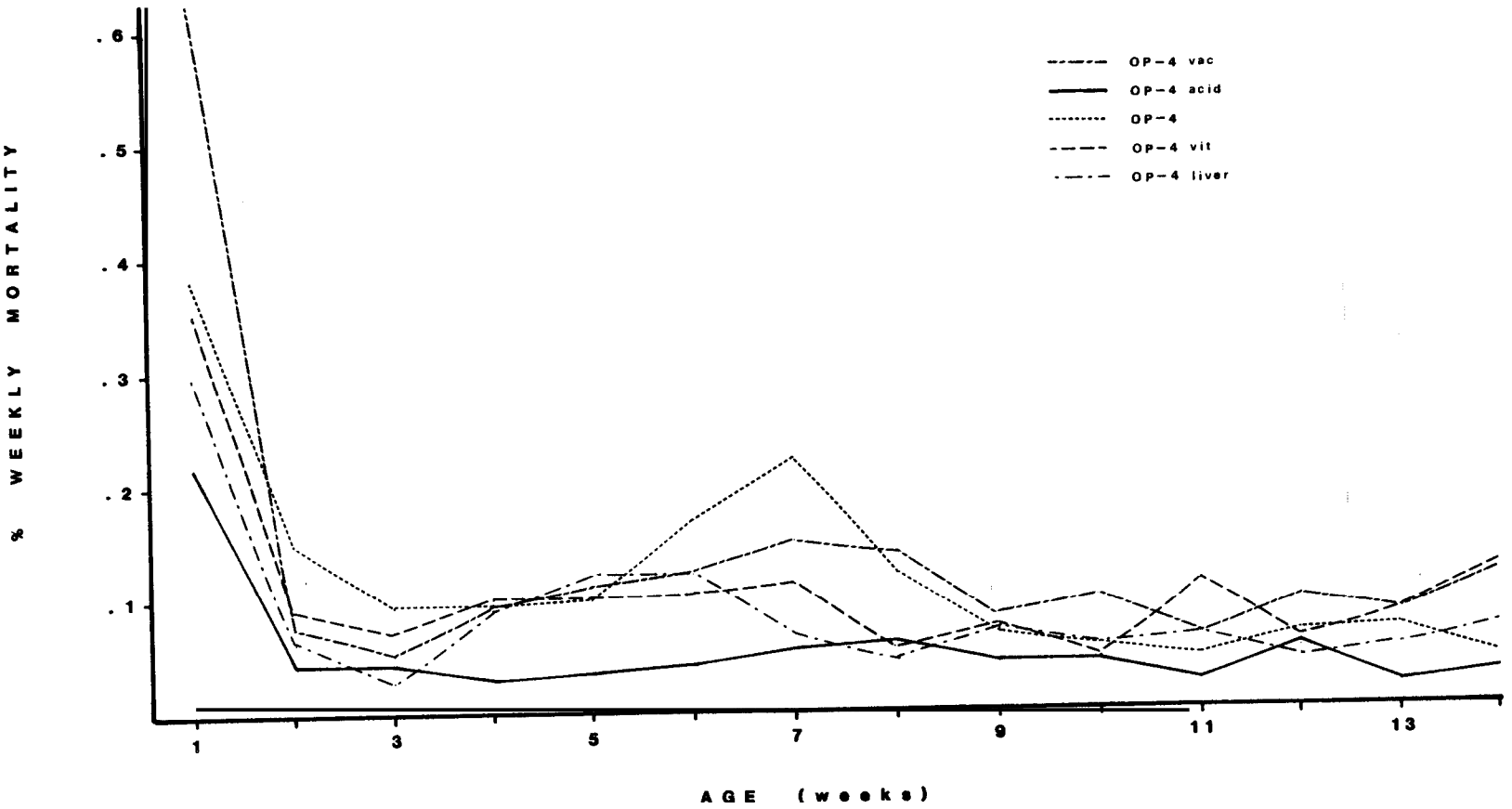


Figure 4. Comparison of mortality with age in test diets.

healthy fish. The most significant histopathological change was proliferation of lamellar epithelium which often started at the proximal end of the filament and occasionally resulted in fusion of several filaments (Fig. 5).

Histologically, tissues of moribund fish exhibited many pathological changes. Livers of fish with ST showed reduced glycogen vaculation and mild diffused karyolysis (nuclear dissolution) of liver cells. Pancreatic acina cells, important in producing digestive enzymes, were often devoid of zymogen granules which are the precursors of digestive enzymes. Such changes may be related to reduced food intake, confirmed by the absence of food, especially in the lower gastro-intestinal tract in many of the fish exhibiting clinical signs of ST. Sloughing of mucosal epithelium of stomach and intestine were seen in several fish, often resulting in the presence of a mucoid plug in the lower intestine. Edema and occasional degeneration of renal tubules were noted in kidneys of a few fish with ST (Charlie Smith, pers. comm.).

Results of vitamin analysis for test diets was inconclusive. Vitamin levels were found to be inconsistent during the storage period (Appendix A). Variable vitamin levels may have been caused by actual variations in vitamin concentrations within each diet or storage/sampling problems during analysis.

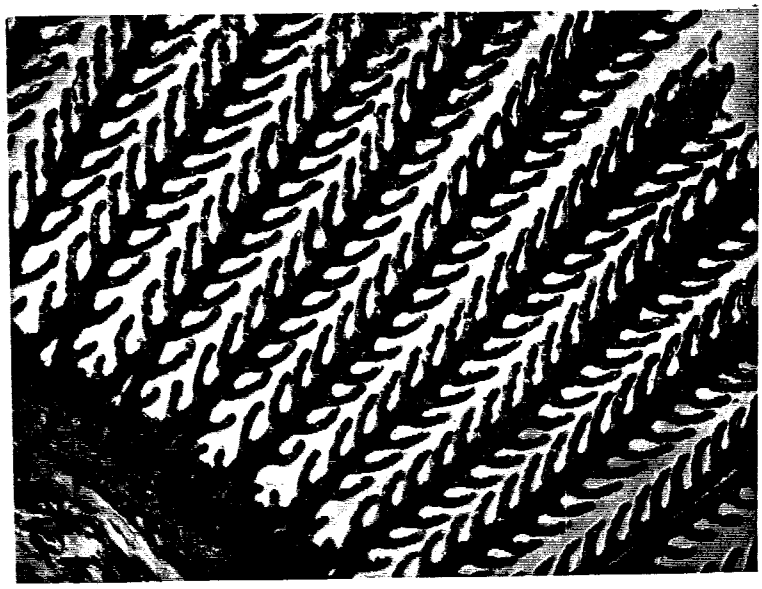
DISCUSSION

During the past four years, investigations into the cause of ST have resulted in substantial progress as to its cause. ST mortalities of production fish have declined from the 24% to 27% in 1980-82 to 9.1% this year (Fig. 6). Studies have indicated this syndrome to be a nutritional problem caused in part by dietary deficiency and the rearing of chinook fry for extended periods in water temperatures of 36-39 F.

Diets tested this year did not prevent ST mortalities but substantially reduced them. The OP-4 acid diet was superior to all others in reduced mortality, growth and body condition. In addition, fish fed OP-4 acid appeared healthier than the other test groups during the entire study.

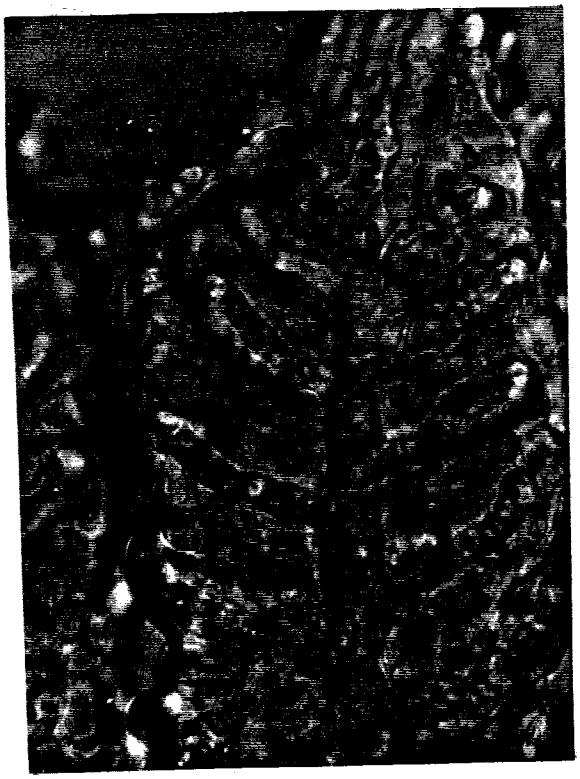
ST mortalities in the 1982 diet study were characterized by a sharp increase in weekly mortalities (Thorpe and Hutchinson 1983). This year, mortalities in all test groups were lower than in past years and there was an absence of that sudden increase. The 1982 study also stated that the springtime increase of our water temperature was correlated with the onset of ST. Increases in water temperatures do not cause the disease, but because of increased fish metabolism, mortalities are accelerated.

Figure 5. Comparison of gills from normal fish and from fish with Spring Thing.



← Histological preparation of a normal gill.

Wet mount of a gill from a fish with Spring Thing. Note the hyperplasia of lamellar epithelium. →



← Wet mount of a gill from a fish with Spring Thing. Note the complete occlusion of interlamellar space.

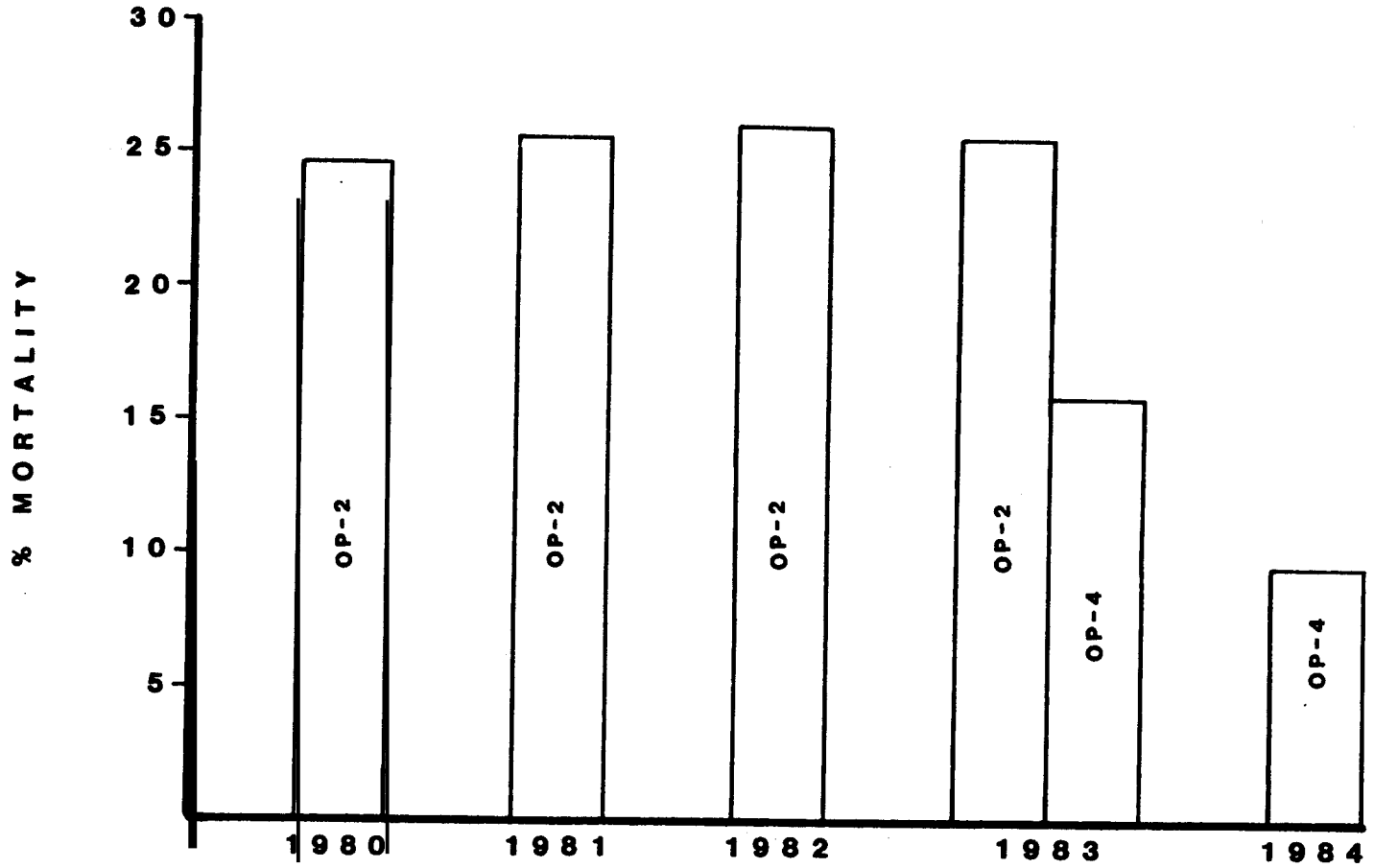


Figure 6. Spring Thing mortalities in production fish with regard to diet, 1980-1984.

Water temperature and quality apparently plays a significant role in this syndrome in that rearing of fry in 36-39 F soft water for nearly five months is a stressor on young fish. Cold water reduces metabolic rate, food intake and may alter nutritional requirements and feeding response. Reduced food intake slows the build-up of the fish's body reserves and uptake of various vitamins and minerals. Diet studies at the Fish Cultural Development Center in Bozeman, Montana, found that brood year '83 SFSR summer chinook fry reared in 50 F soft water and fed diets identical to those used at McCall did not contract ST (Smith and Mueller 1984). Thus the importance of water temperature or quality or both is apparent.

All diets contained nutrient levels in excess of the National Research Council's recommended dietary levels for salmonid fishes. However, supplementation of the commercial OMP diet with the B vitamin, pantothenic acid, apparently has reduced mortalities caused by ST under McCall's rearing conditions.

Histologically, gills of moribund fish exhibited a clubbed-gill pathology similar to nutritional gill disease and typical of changes seen in fish fed diets deficient in pantothenic acid (Charlie Smith, pers. comm.). Tissues of fish with ST showed many pathological changes consistent with reduced food intake. However, further studies need to be conducted to determine if such changes are a response to reduced food intake or are truly a pathological condition of the disease.

Other factors may contribute to this disease. Water quality is a question that has not been thoroughly investigated at McCall yet is an important factor in fish culture. As such, dietary efficiency may be directly correlated with McCall's soft almost sterile water.

RECOMMENDATIONS

Further diet studies testing multiple supplements to the OP-4 diet are needed to determine the relationship of nutrient levels to ST mortalities.

Investigate mineral supplementation of hatchery water and its effect on fish health.

ACKNOWLEDGEMENTS

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We would also like to thank Charlie Smith for his dedicated work on this project. His histopathological expertise has been the key to the success of this five-year investigation.

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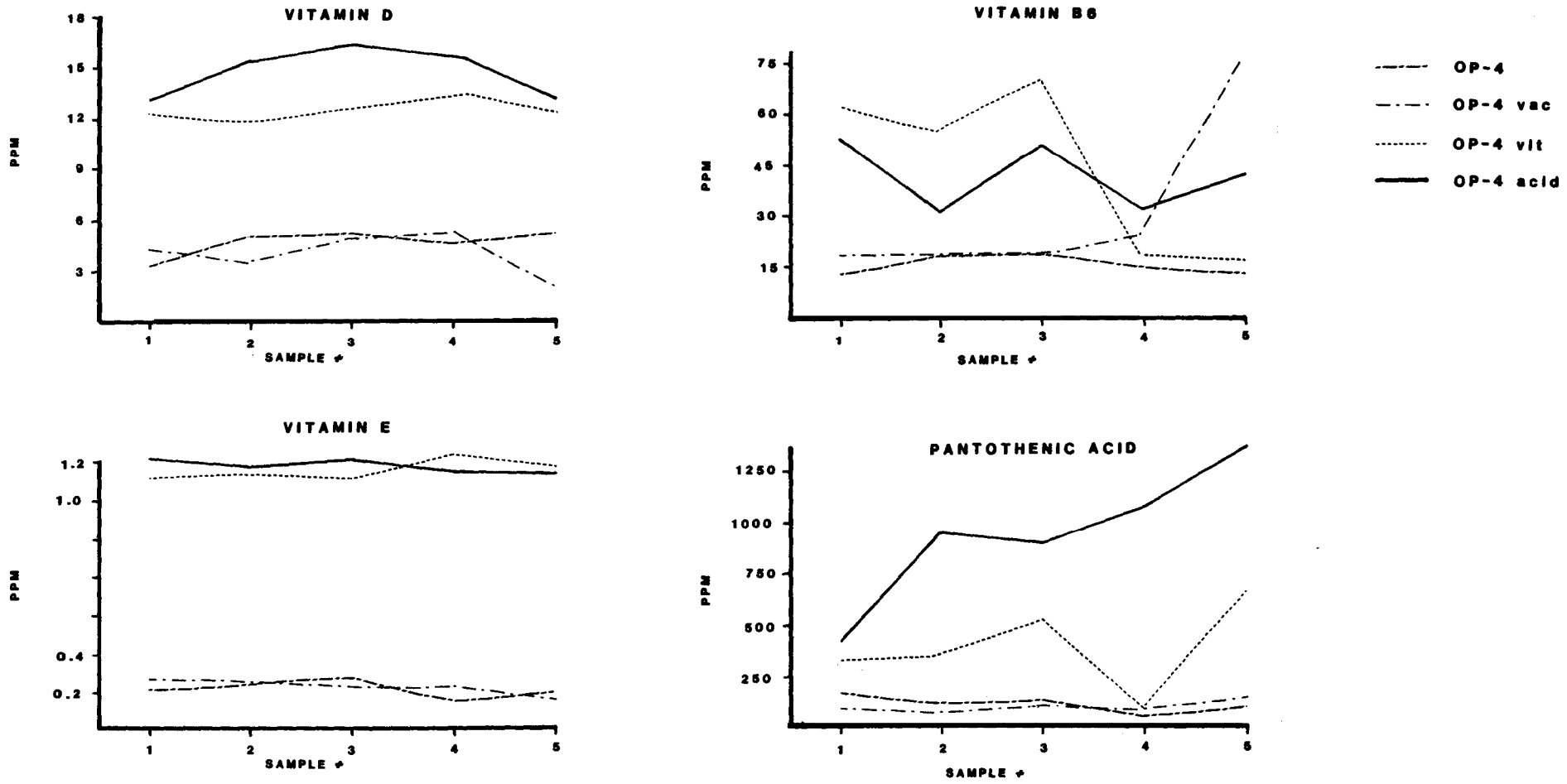
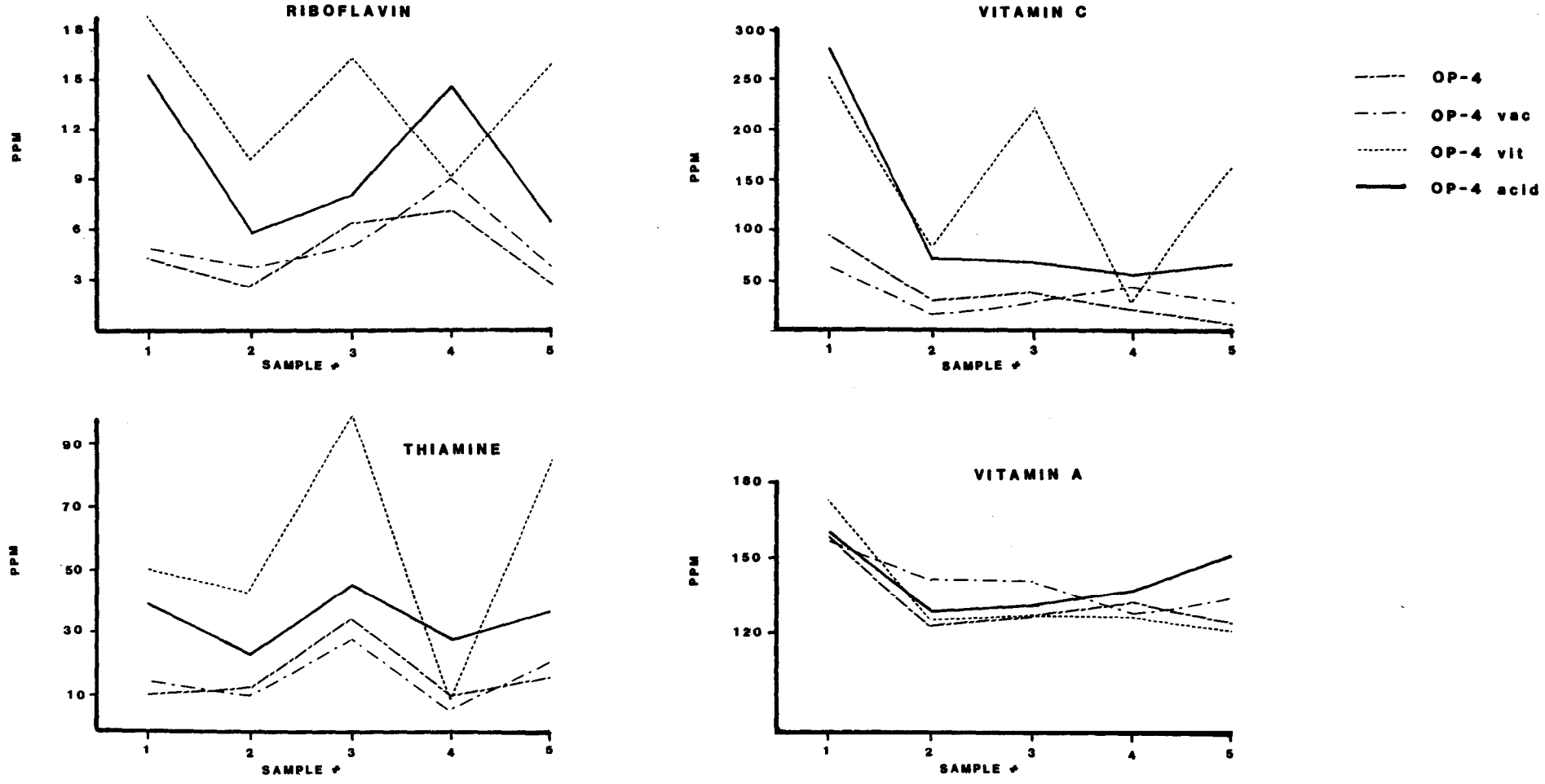
APPENDIX



Appendix A. Results of vitamin analysis of test diets over five sample periods.



Appendix A. (con't).